

U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 23 February 2006

In the Matter of:

HAZEL L. PERKINS
(widow of MOSES EARLY PERKINS, deceased),
Claimant,

v.

Case No.: 2004-BLA-06270

CONSOLIDATION COAL COMPANY,
Employer,

and

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS,
Party-in-Interest

Appearances:	Frederick K. Muth, Esq. Hensley, Muth, Garten & Hayes For the Claimant	Christopher M. Hunter, Esq. Jackson Kelly, PLLC For the Employer
Before:	William S. Colwell Administrative Law Judge	

DECISION AND ORDER – DENYING BENEFITS

INTRODUCTION

This proceeding arises from a claim for survivor's benefits under the Black Lung Benefits Act (the "Act"), 30 U.S.C. §§ 901 *et. seq.* Benefits under the Act are awarded to coal miners who are totally disabled within the meaning of the Act due to pneumoconiosis, or to the survivors of coal miners who were totally disabled due to pneumoconiosis at the time of their deaths (for claims filed prior to January 1, 1982), or whose death was due to pneumoconiosis. Pneumoconiosis, commonly known as black lung, is a dust disease of the lungs resulting from coal dust inhalation. The Act and its implementing regulations define pneumoconiosis as a chronic dust disease of the lungs and its sequelae, including respiratory and pulmonary impairments, arising out of

employment in the Nation's coal mines. 30 U.S.C. § 902(b); 20 C.F.R. § 718.201 (2004). In this case, the Claimant, Hazel L. Perkins, alleges that the Miner's death was due to pneumoconiosis.

The Department of Labor has issued regulations governing the adjudication of claims for benefits arising under the Black Lung Benefits Act at Title 20 of the Code of Federal Regulations. [T]he procedures to be followed and standards applied in filing, processing, adjudicating, and paying claims, are set forth at 20 C.F.R., Part 725, while the standards for determining whether the Miner's death was due to pneumoconiosis are set forth at 20 C.F.R., Part 718.

I conducted a formal hearing on this claim on October 5, 2004 in Pipestem, Virginia. All parties were afforded a full opportunity to present evidence and argument, as provided in the Rules of Practice and Procedure before the Office of Administrative Law Judges. 29 C.F.R. Part 18 (2004). At the hearing, Administrative Law Judge Exhibit ("ALJX") 1, Director's Exhibits ("DX") 1-38, and Employer's Exhibits ("EX") 1-4 were admitted into evidence. The record was held open after the hearing to allow the parties to submit additional argument. The Claimant has submitted a letter brief in support of the claim, and the Employer has also submitted its closing argument. The record is now closed.

In reaching my decision, I have reviewed and considered the entire record pertaining to the claim before me, including all exhibits admitted into evidence, the testimony at hearing, and the arguments of the parties. Some exhibits, while admitted, will not be considered in view of the evidentiary limitations set forth at Section 725.414(a).

PROCEDURAL HISTORY

The Miner, Mr. Moses Early Perkins, filed his first claim for benefits under the Act on November 30, 1982. DX-1. The file in this claim has been lost. On May 3, 1993, Mr. Perkins filed his second claim. DX-2-1. In a Decision and Order Denying Benefits dated January 19, 1995, and filed on January 24, Administrative Law Judge Jeffrey Tureck found that the Miner failed to establish the existence of pneumoconiosis, and thus denied the claim. DX-2.

The Miner died on August 8, 2001. Tr. 17; DX-4, 11. The Claimant filed the instant survivor's claim on May 28, 2002. DX-4. On July 21, 2003, after the initial development of the record, the District Director issued a *Schedule for the Submission of Additional Evidence*. DX-25. 20 C.F.R. § 725.410(a). The District Director also named Consolidation Coal Company as the responsible operator. 20 C.F.R. §§ 725.490 - 725.495. The District Director also concluded that, at that stage in the claim, the Claimant would not be entitled to survivor's benefits. DX-25. On January 21, 2004, after the receipt of additional evidence, the District Director issued a *Proposed Decision and Order - Denial of Benefits*. DX-31. 20 C.F.R. § 725.418. By letter dated January 27, 2004, Claimant's counsel requested a formal hearing. DX-33. On May 5, 2004, this

matter was referred to this Office for a formal hearing. DX-37.

APPLICABLE STANDARDS

Because Claimant filed this application for survivor's benefits after March 31, 1980, the regulations set forth at Part 718 apply. *Saginaw Mining Co. v. Ferda*, 879 F.2d 198, 204, 12 B.L.R. 2-376 (6th Cir.1989). 20 C.F.R. § 718.2. This claim is governed by the law of the United States Court of Appeals for the Fourth Circuit, because the Miner was last employed in the coal industry in the State of West Virginia, within the territorial jurisdiction of that court. DX-6. *Danko v. Director, OWCP*, 846 F.2d 366, 368, 11 B.L.R. 2-157 (6th Cir. 1988). See *Broyles v. Director, OWCP*, 143 F.3d 1348, 1349, 21 B.L.R. 2-369 (10th Cir. 1998); *Kopp v. Director, OWCP*, 877 F.2d 307, 12 B.L.R. 2-299 (4th Cir. 1989); *Shupe v. Director, OWCP*, 12 B.L.R. 1-200 (1989) (*en banc*).

In order to establish entitlement to survivor's benefits in a claim filed on or after January 1, 1982, Claimant must establish that the Miner had pneumoconiosis arising out of coal mine employment and that the Miner's death was due to pneumoconiosis, that pneumoconiosis was a substantially contributing cause or factor leading to the Miner's death, that the Miner's death was caused by complications of pneumoconiosis, or that the Miner had complicated pneumoconiosis. 20 C.F.R. §§ 718.1, 718.202, 718.203, 718.205(c), 718.304. See *Trumbo v. Reading Anthracite Co.*, 17 B.L.R. 1-85 (1993); *Neeley v. Director, OWCP*, 11 B.L.R. 1-85 (1988). Claimant has the burden of proving each element of entitlement to benefits by a preponderance of the evidence. *Director, OWCP v. Greenwich Collieries [Ondecko]*, 512 U.S. 267, 18 B.L.R. 2A-1 (1994), *aff'g. Greenwich Collieries v. Director, OWCP*, 990 F.2d 730, 17 B.L.R. 2-64 (3d Cir. 1993). The failure to prove any requisite element precludes a finding of entitlement. *Anderson v. Valley Camp of Utah, Inc.*, 12 B.L.R. 1-111 (1989); *Perry v. Director, OWCP*, 9 B.L.R. 1-1 (1986) (*en banc*).

ISSUES

The following principal issues are included for adjudication in this survivor's claim:

1. Whether the Miner suffered pneumoconiosis as defined in the Act and the regulations.
2. Whether his pneumoconiosis arose out of coal mine employment.
3. Whether the Miner's death was is due to pneumoconiosis.
4. Whether the Miner was totally disabled, and, if so, whether this total respiratory disability was due to pneumoconiosis.

See DX-36; Tr. 8-9. At the formal hearing, counsel for the employer acknowledged that Consolidation Coal Company is the appropriately named responsible operator. TR-5, 9. The employer also withdrew as contested issues the issues of whether Mr. Perkins had been a miner and whether he had engaged in coal mine employment after 1969. Tr. 8.

The employer also stipulated to 20 years of qualifying coal mine employment, and withdrew its contest of dependency issues. The employer also contests the validity of the Secretary's regulations, as amended. That issue shall be preserved for further litigation.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Claimant's Testimony

The Claimant, Mrs. Hazel L. Perkins, testified briefly at the hearing. She and Mr. Perkins were married on July 1, 1954. She has not remarried since the Miner's death on August 8, 2001. Tr. 17-18. Mrs. Perkins recounted that the Miner had filed a successful claim for occupational lung disease benefits from the State of West Virginia, and pursuant to that claim was receiving a 20-percent silicosis award. Tr. 18. Mr. Perkins retired in 1977. He had been treated for lung disease by a Dr. Jarboe and Dr. Patel, who had been the Miner's treating physician during the final three years of his life. Tr. 19-20.

MEDICAL EVIDENCE

Evidentiary Limitations

At the outset, we must determine how to apply the limitations on the development of the evidence. The employer has challenged the validity of the Secretary's amended regulations. That challenge is denied and the issue thus preserved for further proceedings.

The pertinent limitations on the development of evidence by an employer are set forth in part as follows:

[725.414(a)] (3)(i) The responsible operator designated pursuant to § 725.410 shall be entitled to obtain and submit, in support of its affirmative case, no more than two chest X-ray interpretations, the results of no more than two pulmonary function tests, the results of no more than two arterial blood gas studies, no more than one report of an autopsy, no more than one report of each biopsy, and no more than two medical reports. Any chest X-ray interpretations, pulmonary function test results, blood gas studies, autopsy report, biopsy report, and physicians' opinions that appear in a medical report must each be admissible under this paragraph or paragraph (a)(4) of this section.

* * *

(ii) The responsible operator shall be entitled to submit, in rebuttal of the case presented by the claimant, no more than one physician's interpretation of each chest X-ray, pulmonary function test, arterial blood gas study, autopsy or biopsy submitted by the claimant under paragraph

(a)(2)(i) of this section and by the Director pursuant to § 725.406. In any case in which the claimant has submitted the results of other testing pursuant to § 718.107, the responsible operator shall be entitled to submit one physician's assessment of each piece of such evidence in rebuttal. In addition, where the claimant has submitted rebuttal evidence under paragraph (a)(2)(ii) of this section, the responsible operator shall be entitled to submit an additional statement from the physician who originally interpreted the chest X-ray or administered the objective testing. Where the rebuttal evidence tends to undermine the conclusion of a physician who prepared a medical report submitted by the responsible operator, the responsible operator shall be entitled to submit an additional statement from the physician who prepared the medical report explaining his conclusion in light of the rebuttal evidence.

* * *

[725.414(a)] (4) Notwithstanding the limitations in paragraphs (a)(2) and (a)(3) of this section, any record of a miner's hospitalization for a respiratory or pulmonary or related disease, or medical treatment for a respiratory or pulmonary or related disease, may be received into evidence.

* * *

[725.414] (c) *Testimony.* A physician who prepared a medical report admitted under this section may testify with respect to the claim at any formal hearing conducted in accordance with subpart F of this part, or by deposition. If a party has submitted fewer than two medical reports as part of that party's affirmative case under this section, a physician who did not prepare a medical report may testify in lieu of such a medical report. The testimony of such a physician shall be considered a medical report for purposes of the limitations provided by this section. A party may offer the testimony of no more than two physicians under the provisions of this section unless the adjudication officer finds good cause under paragraph (b)(1) of § 725.456 of this part.

20 C.F.R. §§ 725.414(a), (c).

Section 725.456(b)(1) permits the introduction of evidence beyond the limits set forth in Section 725.414(a) for "good cause." As was stated by the United States District Court for the District of Columbia in ruling on a challenge to the Secretary's rule amendments, including the evidentiary constraints:

While the rules limit documentary medical evidence, the DOL incorporated a "good cause" exception, see 20 C.F.R. § 725.456(b)(1), as a procedural

safeguard for cases where the ALJ *needs additional evidence to make an adequate determination of the claimant's eligibility.*

National Mining Ass'n v. Chao, 160 F.Supp. 2d 47, 79 (D.D.C. 2001) (emphasis added), *aff'd in part, rev'd in part*, *National Mining Ass'n v. U. S. Dept. of Labor*, 292 F.3d 849 (D.C.Cir. 2002).

In order to render an “adequate determination” of Claimant’s entitlement, I find “good cause” to admit the record from the second Miner’s claim. DX-2. I find the evidence in the Survivor’s claim to be so intertwined with significant evidence in the Miner’s claim that I need to review the evidence in both cases to best provide a balanced decision in the Survivor claim. I find some of the most forthright medical opinions regarding the issue of pneumoconiosis are in the Miner’s claim.

Although it has been formally admitted, I shall not consider the report from Dr. Caffrey. DX-23. The employer posits that this report constitutes evidence in “rebuttal” to the biopsy reports that otherwise appear in the record. I conclude that Dr. Caffrey’s report does not constitute proper rebuttal evidence.

The evidentiary limitations permit the introduction of evidence in rebuttal to evidence submitted by a claimant pursuant to Section 725.414(a)(2)(i) or by the Director pursuant to Section 725.406. The medical opinions from Dr. Caffrey do not qualify as proper rebuttal to evidence submitted pursuant to these provisions. In the alternative, given the thorough opinions from Dr. Bush, I find that Dr. Caffrey’s conclusions regarding biopsy evidence are cumulative.

Death Certificate

The Miner, as noted above, died on August 8, 2001 at the age of 70 years. DX-11. The death certificate listed the cause of death as “acute respiratory failure” due to, or as a consequence of, chronic obstructive pulmonary disease. The death certificate was signed by Dr. Vishnu Patel, who also listed “pneumoconiosis” as another significant condition that contributed to Mr. Perkins’s death. DX-11.

Record from Miner’s Claim

Without adopting earlier findings and conclusions by prior adjudicators, I do incorporate by reference those lists of exhibits and evidence as previously set forth. DX-2. *See generally, Wheeler v. Apfel*, 224 F.3d 891, 895 n. 3 (8th Cir. 2000). Notwithstanding, any prior evidence, whether or not specifically set forth herein, has been evaluated *de novo*.

Treatment Records

Princeton Community Hospital

The record includes treatment documents from the Princeton Community Hospital. DX-13, 15. The records show that he had lengthy stays at this hospital from 1999 until his death.

Mr. Perkins entered the hospital on November 13, 1999 complaining of chest pain. Dr. Patel's admission impression indicated "chest pain, rule out cardiac ischemia, rule out myocardial infarction," "bronchospasm, rule out acute COPD," "history of lung carcinoma," and "chronic chest pain syndrome." The discharge summary showed a diagnosis of chest pain, with secondary diagnoses of bronchospasm, history of lung cancer and chronic pain syndrome.

After Mr. Perkins was discharged after a December 6, 1999 admission, the discharge summary noted in the social history that he "did not work in the coal mines." The past medical history consisted of "advanced COPD with obstructive lung disease," lung cancer, status post MI and "CHF and mild anemia." The discharge summary reflected a primary diagnosis of an "interstitial perforation," with secondary diagnoses of "chronic obstructive lung disease" and "chronic obstructive pulmonary disease," "history of lung carcinoma" and "coronary artery disease." A diagnostic laparoscopy was conducted during this admission.

A pulmonary function test conducted on February 2, 2000, produced results consistent with severe airway obstruction.

He was admitted on February 11, 2000 for chest pain. The principal diagnosis was chest pain, "most probably chronic in nature." The secondary diagnoses included an acute exacerbation of chronic obstructive pulmonary disease, "resolving." He also suffered from hypoxemia, history of lung cancer, chronic pain syndrome. The discharge summary from an admission for January 26, 2000 noted complaints of chest pain. It was thought at that time that the chest pain could be related to coronary artery disease. Mr. Perkins also suffered from severe end-stage chronic obstructive pulmonary disease, history of hypoxemia and history of lung cancer.

Mr. Perkins was admitted on February 11, 2000 for chest pain. It was noted that he carried diagnoses of severe end-stage COPD and lung cancer and heart disease (CAD & CHF). The discharge summary concluded that he suffered from "bronchorrhea." The principal diagnosis of "end-stage COPD" followed an admission on February 28, 2000.

On numerous occasions in 2001, Mr. Perkins was treated at the Princeton Community Hospital for recurrent respiratory failure with a series of "therapeutic bronchoscopy" procedures with "bronchoalveolar lavage" to relieve mucous plugging. These procedures were intended to relieve "poor tracheobronchial clearance leading to

mucous plugging and respiratory failure.” EX-13, 15.

Drs. Prescott and Mohammed Dawood each prepared consultation reports during this hospital stay. The latter saw Mr. Perkins for “hyperkalemia,” noting a history “[s]ignificant for COPD, hypertension and coronary artery disease[.]” Dr. Prescott also noted a “long history of COPD and coronary artery disease.”

Mr. Perkins was admitted on February 6, 2001 with worsening shortness of breath and chest pain. Dr. Patel’s principal diagnosis was “acute respiratory insufficiency, improving.” Secondary diagnoses included “history of severe chronic obstructive pulmonary disease,” “chronic hypoxemia,” “coronary artery disease,” “myocardial infarction,” and “questionable lung nodule.” Dr. Robert Snidow’s emergency room note indicated that the Miner “is known to have severe endstage COPD with frequent hospitalizations[.]” Dr. Snidow’s impressions were “acute respiratory distress,” “severe hypoxemia with COPD,” “chest pain,” “hypokalemia/electrolyte imbalance” and “anemia.”

After an admission on February 15, 2001 for worsening shortness of breath and chest pain, Dr. Patel thought that the complaints were due to acute respiratory insufficiency “secondary to very poor tracheobronchial clearance with persistent pneumonitis.” The “admission impression” also included pneumoconiosis, chronic obstructive pulmonary disease, lung cancer, coronary artery disease and chronic chest pain syndrome. The principal discharge diagnosis was “pseudomonas pneumonia,” and the secondary diagnoses included pneumoconiosis. Dr. Al-Attar consulted, and reported that the Miner suffered from COPD with a history of “persistent shortness of breath secondary to poor clearing of bronchial secretions and who now has pseudomonas aeruginosa and pneumonia.”

Mr. Perkins entered the hospital on March 15, 2001 complaining of chest pain and shortness of breath. Dr. Prescott consulted in the treatment and reported that the chest pain was “atypical” for coronary artery disease, and thought that the Miner suffered from chronic obstructive pulmonary disease and tachycardia. Dr. Patel’s “admission impression” was that Mr. Perkins suffered from

[a]cute chest pain with a history of coronary artery disease and myocardial infarctions, new myocardial infarction needs to be ruled out. ... Acute respiratory insufficiency secondary to persistent recurrent pneumonitis. Severe COPD ... bronchospasm.

The principal discharge diagnosis was “acute respiratory insufficiency, improving.” Secondary diagnoses were “bronchospasm,” “persistent pneumonitis,” severe “COPD,” hypoxemia, congestive heart failure and rectal discharge.

Mr. Perkins was admitted on April 2, 2001 for acute respiratory failure and was treated with IV antibiotics, steroids, nebulized bronchodilator therapy with clinical improvement.” The principal discharge diagnosis by Dr. Patel was “acute respiratory

insufficiency, improved[.]" with secondary diagnoses of "bronchospasm" and "COPD."

Mr. Perkins was admitted on April 12, 2001 with chest pain, worsening shortness of breath and lung congestion. Dr. Patel's discharge summary noted in the "admission impression" portion of the summary that the Miner's "[c]hest pain is mostly pulmonary in origin ... [and that r]ecurrent respiratory failure secondary to very poor tracheal bronchial clearance causing persistent mucous plugging." The principal discharge diagnosis was "acute respiratory insufficiency, improved[.]" with secondary diagnoses of "bronchospasm" and "COPD."

Mr. Perkins was admitted on April 23, 2001. In the May 1 discharge summary, Dr. Patel noted that the Miner "carr[ie]d the diagnosis of severe end-stage COPD[.]" Dr. Gordon Prescott saw the Miner for coronary artery disease and bradycardia. The discharge summary noted a principal diagnosis of "acute bradycardia, most probably cardiac in origin[.]" with secondary diagnoses of "anemia," "hyperkalemia" and "respiratory failure."

Mr. Perkins was admitted on May 3, 2001 with complaints of respiratory distress, expectoration, loss of appetite and hematuria. Dr. Patel related on the discharge summary on May 15, 2001 that the Miner "carr[ie]d the diagnosis of severe end-stage COPD, CA of the lung, lobectomy, pneumoconiosis, CHF[.]" The principal discharge diagnosis was "acute chest pain, myocardial infarction was ruled out." Secondary diagnoses were "acute respiratory insufficiency," "bronchospasm" and "chronic hypoxemia."

Dr. Inas Al-Attar examined the Miner during this May 3 admission. He described the Miner as one "known to have severe end-stage COPD with chronic hypoxia, chronic chest pain, coronary artery disease, and a history of lung CA ... who has had numerous hospitalizations due to recurrent respiratory symptoms secondary to poor clearing of secretions from his bronchial tree." Dr. Al-Attar noted that Mr. Perkins's "respiratory distress" was "gradually improving on Imipenem, Zithromax, and steroids." Dr. Gordon F. Prescott examined the Miner during the May 3 hospitalization, and noted that he had a "long history of CAD and end stage COPD."

Dr. Inas Al-Attar examined the Miner during his May 15 admission, having been asked to provide a consultation because of "persistent pneumonia." Dr. Al-Attar noted that the Miner was "known to have end stage COPD with chronic hypoxia, chronic chest pain, coronary artery disease and a history of lung CA[.]" Dr. Al-Attar's impression was that the Miner had "end stage chronic obstructive pulmonary disease (COPD) and persistent bronchopneumonia which is most likely due to pseudomonas aeruginosa. The last organism cultured was resistant to antibiotics tested except for tobramycin."

Dr. Hatahet was consulted, and he examined the Miner during this admission. He authored an "Interim Note," which reflects his thorough review of the Miner's chart because Dr. Hatahet was not familiar with Mr. Perkins. He wrote that "[s]ubjectively, the patient has been complaining of dyspnea constantly." Auscultation of the chest on

examination revealed “very harsh breath sounds ... [and] diffuse loud rhonchi.” Dr. Hatahet reported that heart sounds were difficult to hear because of “respiratory interference.” Dr. Hatahet offered the following impression:

1. Acute respiratory failure.
2. Decompensated chronic respiratory insufficiency.
3. Terminal emphysema.
4. Recurrent atelectasis in the right lung, most likely related to:
5. Extremely viscid sputum. ...
6. Bronchogenic carcinoma.
7. Multiply resistant gram negative pneumonia.

An “interim note” was prepared by Dr. Yasir Hatahet after a consultation. Dr. Hatahet reported on August 5 that an examination of the chest revealed “[d]iffuse loud rhonchi,” and “harsh breath sounds.” His impressions were:

1. Acute respiratory failure.
2. Decompensated chronic respiratory insufficiency.
3. Terminal emphysema.
4. Recurrent atelectasis in the right lung, most likely related to:
5. Extremely viscid sputum. Frequent administration of promethazine might have added to sputum viscosity.
6. Bronchogenic carcinoma, post resection and radiation.
Multiply resistant negative pneumonia.

On August 8, 2001, after he was again in respiratory distress, the Miner’s family decided on palliative care – Mr. Perkins chose not to be placed on a ventilator. He went into respiratory arrest and expired. Dr. Patel’s principal diagnosis was “acute respiratory failure,” with secondary diagnoses of

1. Pneumoconiosis.
2. Chronic obstructive pulmonary disease.
3. Hypoxemia.
4. Coronary artery disease.
5. Cancer of the lungs.
6. History of congestive heart failure. Recurrent pneumonitis.

Princeton Internists

Medical records from the Princeton Internists medical practice indicate that the Miner treated with physicians in that group from the 1970s. DX-14. Office notes from 1976 were compiled by Drs. Piracha, Prescott and Javed.

The office notes that are submitted for the survivor's claim begin on January 3, 1976.¹ The more recent notes said that the Miner was evaluated for blood pressure, and that he smoked "one pack per day." On February 14, 1976, the notes reflect that Mr. Perkins was interested in applying for "black lung." Notes from August 14, 1976 show a concern for the black lung claim, and that the chest was stable and clear.

On April 27, 1979, the Miner complained of shortness of breath on exertion. Notes from November 18, 1980, reflect that the Miner wanted "some black lung papers filled out." He complained of chest pain and was taking Aldoril. On October 30, 1981, Mr. Perkins began taking Lopressor in addition to the Alderol.

On March 29, 1982, Mr. Perkins suffered some chest pain for two days with associated shortness of breath. He was taking Nitroglycerin in addition to the other medications. On April 12, 1982, the drug Inderal replaced the Lopressor. The blood pressure had climbed on August 2, 1982, and Mr. Perkins received a refill of Ativan, which he took along with the Aldoril and Inderal. Notes from November 4, 1982 reflect that "he has a history of hypertension and back injuries." The doctor also recorded that "[h]e says he smokes about 1-2 packs per day[.]"

On December 6, 1983, Mr. Perkins complained of "[s]ome shortness of breath." On physical examination, the "chest was generally clear." "Lungs are clear" on May 24, 1984. The notes dated April 22, 1986 indicate that the Miner was continuing to have problems with his back, and that he "finally got compensation."

The Miner's "chest [was] clear" on examination on August 7, 1987. On August 14, he went to the emergency room for chest pain. "Lung scans performed were normal." He improved after receiving Wygesic, and the chest was clear on examination. On September 18, 1987, he complained of chest congestion. An examination detected "some rhonchi bilaterally." The chest was clear, however, on December 8, 1987 and January 12, 1988, while a "rhonchi bilaterally" were observed on June 29, 1988, and the chest was "clear" as reflected in notes from October 5, 1988. He continued on Inderal, Aldoril and Vasotec.

Mr. Perkins's complaints of a nonproductive cough and chest pain were recorded on February 7, 1989. An antibiotic was prescribed on this date.

Some rhonchi were detected as shown in notes dated June 20, 1989. On November 17, 1989, the notes indicate that "[h]e had a non productive cough that he is unable to get rid of despite Erythromycin given to him by another physician." Rhonchi were observed on chest examination.

Although Rhonchi were detected on January 15, 1990, there was no wheezing and lungs were clear the following March. Notes from July 24, 1990 show that the Miner denied any shortness of breath, although the October 30 entry shows the

¹ Those offered for the Miner's claim commenced in the early 1970s. DX-2.

presence of “some rhonchi bilaterally. NO wheezes.”

Lungs were clear as reflected in notes for January 30, 1991. Notes from May 3, 1991 show a variety of medications. On June 14, 1991, the notes indicate that the Miner complained of a cough that produced “small amounts of pur[u]lent sputum. Lungs demonstrate some rhonchi bilaterally. No wheezes and no rales are evident.” August 20, 1991 notes reflect a complaint of shortness of breath. On March 9, 1992, Mr. Perkins was “a little more short of breath than he [had] been.” He had a non-productive cough, the lungs demonstrated some rhonchi and no wheezing.

The notes from April 15, 1992 show that Dr. Jabour, who was seeing Mr. Perkins for pulmonary problems, thought that the Miner had “acute bronchitis.” The Miner was “breathing fairly well” by May 19. Mr. Perkins suffered a seizure in November, 1992.

The Miner was treated for pneumonia, and by January 11, 1993 had been breathing well. Bilateral rhonchi were observed, and good breathing was also observed as noted on March 18. A chest x-ray showed some atelectasis, according to notes from April 19, 1993. Lungs also showed a few rhonchi as noted on May 12 and July 13, and December 16, 1993.

Mr. Perkins complained of some “short[ness] of breath at time” on April 13, 1994, but reported on June 24, 1994 that he had been “breathing fairly well.” Notes from September and December, 1994, and February, 1995, reflect some bilateral rhonchi.

Notes from May 23 and September 12, 1995 reflect clear lungs, and rhonchi were reported in December, 1995 and March and June, 1996. The diagnosis of lung cancer was noted on November 13, 1996. Radiation therapy was conducted and the entry for January 14, 1997 stated that Mr. Perkins had finished a course of this treatment. Notes from July 24, 1997 report that the Miner had been hospitalized for pneumonia and shortness of breath. He was receiving antibiotics from Dr. Jabour, and an examination detected “some rhonchi.”

By August 25, 1997, the Miner had been using oxygen. Notes from December indicate that he had a chronic cough that produced grey sputum at times. A CT scan was scheduled for February 18, 1998. Mr. Perkins was taking Prednizone. He complained in October, 1998 that he was still short of breath, and an examination revealed shortness of breath.

On January 12, 1999, an examination revealed some rhonchi at both bases with no wheezes or rales. Similar chest examination results were observed in August and December, 1999. Notes from December report a productive cough. The final entry, made on August 15, 2000, notes that “dense rhonchi through his left chest” were observed, along with chest pain thought to be related his breathing.

Chest X-Rays

The newly submitted evidence for the survivor's claim includes the following chest x-ray interpretations:

Ex. No.	X-Ray Date Reading Date	Physician	Credentials	Interpretation
DX-14	04-24-98 04-24-98	Ahmed	B/BCR DX-2 [CX-3]	Unclassified "no new infiltrates ... changes [in] right hilum ... could be post therapy ..."
DX-14	11-12-96 11-12-96	Rahman		unclassified "no focal infiltrate ... no acute pneumonia or CHF."
DX-14	04-17-98 04-17-98	Rahman		unclassified "left lung is clear ... complete opacification of the right hemithorax[.]"
DX-14	11-19-99 11-19-99	Ahmed	B/BCR	unclassified "underlying COPD. Changes could be post-radiotherapy."
DX-14	12-20-99 12-20-99	Groten		unclassified "no new focal infiltrate is identified[.]"
DX-13	11-04-99 11-04-99	Patel		"Matching ventilation and perfusion lung scans with right upper lobe defect corresponding to post surgical and post radiation therapy changes of the right upper lobe[.]"
DX-13	11-15-99 11-15-99	Patel		unclassified "post surgical and post radiotherapy change in the right chest."
DX-13	02-06-01 02-06-01	Ahmed	B/BCR	unclassified "Question of a 1 cm nodule ... in this patient with previous lung cancer. No new infiltrate. ... cardiomegaly."
DX-13	02-15-01 02-15-01	Shahan		unclassified "postsurgical and probably post radiotherapy change in the right upper chest. No acute disease[.]"
DX-13	03-02-01 03-02-01	Ahmed	B/BCR	unclassified "status post right thoracotomy, scarring and loss of volume right lung. Borderline to mild cardiomegaly and COPD."
DX-13	03-06-0[1] 03-06-0[1]	Groten		unclassified "postoperative changes ... no new focal infiltrate ... "

DX-13	03-11-01 03-11-01	Aycoth	B/BCR DX-2 [CX-2]	unclassified "Post thoracotomy changes of right upper lung zone with suspicious right suprahilar infiltrate."
DX-13, 15	03-15-01 03-15-01	Groten		unclassified "postsurgical change ... previously described speculated density in the right suprahilar region with associated increased density in the right lung apex is again noted ..."
DX-13, 15	03-17-01 03-17-01	Olson		unclassified "post right thoracotomy sequelae evidently related to previous therapy for lung cancer ... no definite superimposed consolidated infiltrate or CHF"
DX-13	04-02-01 04-02-01	Pathak	DX-2 [CX-6]	unclassified "right thoracotomy sequelae are noted unchanged ... left lung remains clear."
DX-13	04-11-01 04-11-01	Cappiello	B/BCR [CX-5]	unclassified "Persistent cardiomegaly without gross CHF or active infiltrate ... scarring in the right lung Change of COPD."
DX-13	04-23-01 04-23-01	Groten		unclassified "... postoperative change and volume loss of the right hemithorax. No new focal infiltrate is seen."
DX-13	05-03-01 05-03-01	Aycoth	B/BCR	unclassified "No new focal infiltrates seen and ... fibrotic density scarring of right suprahilar lung zone."
DX-13	05-05-01 05-05-01	Ahmed	B/BCR	unclassified "scarring right lung ... question of a right perihilar pneumonic infiltrate."
DX-24	05-05-01 02-24-03	Wiot	B/BCR	no evidence of pneumoconiosis
DX-13	05-10-01 05-10-01	Shahan		unclassified "scarring and volume loss ... no acute disease"
DX-13	05-20-01 05-20-01	Groten		unclassified "... extensive scarring involving the right hilum and right lung apex."
DX-13	05-25-01 05-25-01	Groten		unclassified "... scarring involving the right lung apex and right hilum."
DX-30	05-31-01 11-13-03	Aycoth	B/BCR	unreadable for presence or absence of pneumoconiosis

DX-13	06-09-01 06-09-01	Shahan		unclassified "Borderline cardiomegaly, increased. Probable post-radiotherapy scarring ..."
DX-13	06-24-01 06-24-01	Ahmed	B/BCR	unclassified "Scarring in the right upper lung, which could be affect of previous surgery and possible radiation changes ... No new infiltrates or nodules in the lung fields ... Scarring and loss of volume ..."
DX-13	07-11-01 07-11-01	Cappiello	B/BCR	Unclassified. "Residues of previous right thoracotomy with pleural parenchymal scarring ... No evidence of infiltrate or CHF"
DX-13	07-31-01 07-31-01	Rahman		Unclassified. "Persistent right hilar opacity and right upper lobe fibrosis, unchanged" "Recurrent disease cannot be excluded"
DX-30	07-31-01 11-13-03	Aycoth	B/BCR	unreadable for presence or absence of pneumoconiosis
DX-24	07-31-01 02-24-03	Wiot	B/BCR	no evidence of pneumoconiosis

Biopsy and Pathology Evidence

Dr. Jabour conducted a bronchoscopy on April 11, 1998. DX-14. The postoperative diagnoses were "pneumonia, pulmonary infiltrate, possible bronchogenic carcinoma versus chronic pneumonitis."

A lower lobe lung biopsy was conducted at the Princeton Community Hospital on April 20, 1998. Dr. Pardasani diagnosed "alveolar cell hyperplasia." DX-14.

A transbronchial biopsy was conducted on November 13, 1999. The post-operative diagnosis was "persistent expectorations with a history of CA ... rule out recurrence of the CA of the lung." DX-13.

A biopsy conducted on November 19, 1999 by Dr. F. Pia at this hospital revealed sections described as showing "bronchial tissue covered by respiratory type epithelium" and "stroma [consisting of] fibrocollagenous tissue." DXs-13, 14.

A bronchoscopy conducted on January 11, 2000 showed "persistent shortness of breath with chest pain with persistent expectoration, rule out endobronchial lesion – history of lung CA." DX-13.

A biopsy of the right lower lung conducted on January 18, 2000, studied specimens from the right lower pulmonary lobe. "No significant abnormality [was]

noted.” DX-13.

A cytology study conducted on February 18, 2000 was “negative for malignant cells.” DX-13.

Dr. Pia examined a bronchus tissue on May21, 2001. DX-15. This tissue was “negative for malignant cells.” The clinical diagnosis was “recurrent pulmonary failure.”

The employer secured the consultation opinion of pathologist Dr. Stephen T. Bush. DX-16. Dr. Bush is board-certified in anatomic and clinical pathology. EX-4 at 8. In his January 13, 2003, report, Dr. Bush’s review included the findings by the West Virginia Occupational Pneumoconiosis Board, a number of surgical pathology reports from the Princeton Community Hospital, admissions records, the death certificate, imaging studies, Dr. Caffrey’s report and 15 histologic slides.

I shall confine my discussion of Dr. Bush’s review of the biopsy material. I conclude that Dr. Bush’s report otherwise does not constitute appropriate “rebuttal” evidence when he offers an opinion based on records other than the pathology material submitted for his review.

Turning to the slides, Dr. Bush concluded:

1. The lungs show no evidence of coal workers’ pneumoconiosis. The needle biopsy of the lung and the resected lung tissue of 1996 show adenocarcinoma, broncho-alveolar type, with a fibrous and chronic inflammatory reaction to the carcinoma. Four (4) of the lung slides show significant amounts of parenchyma with minimal quantities of black dust pigment consistent with coal dust beneath the pleura. Polarized light examination shows scattered silica and silicate particles associated with the dust. No fibrosis is present due to the dust deposition. The carcinoma is subpleural in location. A lymph node contains metastatic carcinoma and a small amount of black dust is present in some lymph nodes with a localized area containing a moderate number of silica and silicate birefringent particles.

Subsequent biopsies show smaller fragments of lung tissue, some of which show a minimal amount of dust pigment but most free of dust pigment. The bronchial biopsy of the right lower lobe shows bronchial wall cartilage and a bronchial gland with marked mucous cell hyperplasia.

Within the carcinoma and its fibrous reaction is a focus of black dust pigment with a few birefringent silica and silicate particles which appears incidental to the carcinoma. No other dust collections are present in the rest of the lung tissue.

2. Coal workers’ pneumoconiosis was not present[.]

Dr. Bush observed that sufficient lung tissue was presented from the 1996 resected lung specimens to evaluate for the presence of coal workers' pneumoconiosis. He also noted that "[t]he amount of carbon dust particles in the lung was minimal as was the amount of dust in the lymph nodes, where dust tends to be concentrated." He opined that "[t]hese findings are strong evidence against the diagnosis of coal workers' pneumoconiosis in Mr. Perkins." DX-16. Dr. Bush is board-certified in clinical and anatomic pathology. DX-16.

Medical Reports

Dr. David M. Rosenberg

At the request of the employer, Dr. Rosenberg conducted a review of the Miner's medical records and submitted a medical report dated August 9, 2004. EX-1. Dr. Rosenberg, who is board-certified in internal medicine, pulmonary disease and occupational medicine, is currently an Assistant Clinical Professor at the Case Western Reserve University School of Medicine. He is also a B-reader. EX-1.

In his review of prior claim files and employment history, Dr. Rosenberg noted that Mr. Perkins was injured on the job in 1977, suffering a back injury that prevented him from walking up or down steps. The Miner had last worked in the mines as a precision mason. This work entailed heavy lifting. The records also reflect that Mr. Perkins said that he had also smoked cigarettes from 1947 until 1980 at the rate of one-half to three-quarters of a pack per day. Dr. Rosenberg noted, however, that Princeton Hospital records note a more extensive smoking history of one to two packs per day.

Following an extensive review of the medical records, Dr. Rosenberg concluded:

In SUMMARY, at the time of Mr. Perkins' death, he was 70 years of age. He had a long smoking history, and also, had a history of coronary artery disease, having had a myocardial infarction and numerous episodes of unstable angina. He also has chronic back problems and 22 years of coal mining employment. His pulmonary function tests revealed variable obstruction, which improved to a level of mild obstruction. His diffusing capacities corrected for lung volumes were normal, and he did not have restriction; air trapping was noted. Blood gas studies indicated that he did not desaturate with exercise. He was also determined through his lifetime to have bronchoalveolar carcinoma of the lung, and underwent a lung resection; he was noted to have metastatic disease. During the latter stages of his life, he developed recurrent respiratory infections, superimposed on his lung resection and radiation which he received. One should also appreciate, that his chest X-rays were interpreted by the majority of B readers as being negative for the presence of CWP and, pathologically, he was not found to have a pneumoconiosis.

DISCUSSION: Based on a review of the above information, it can be appreciated, that Mr. Perkins did not reveal micronodularity, based on the number and experience of the B readers having reviewed his X-rays. This was confirmed by the CAT scan findings described in the file. It should be noted, a CAT scan is a much more sensitive indicator (compared to the X-ray) for determining the presence of micronodularity. The roentgenographic absence of micronodularity was confirmed pathologically. In association with the absence of CWP, Mr. Perkins had no evidence of restriction, displaying a normal TLC. Also, his diffusing capacity measurements were normal. Consequently, when all the above information is looked at in total, Mr. Perkins did not have the interstitial form of coal workers' pneumoconiosis (CWP).

* * *

There is no question, that coal mine dust exposure can cause airflow obstruction. When this occurs, the coal macule which develops in the terminal bronchioles is associated with the development of focal emphysema [journal citation omitted]. As the macule evolves into micronodular, macronodular disease and potentially complicated CWP, the associated COPD can also progress. [citations to various journals and discussion of studies omitted]...

With respect to Mr. Perkins, he had mild to moderate airflow obstruction with marked air trapping. It is this air trapping which reduced Mr. Perkins' FVC measurement raising his FEV1% values. This overall pattern of airflow obstruction in Mr. Perkins, is not consistent with coal mine dust induced airflow obstruction. Undoubtedly, it related to his long smoking history, the factor which also caused his lung cancer.

With respect to his death, he developed increasing respiratory infections, superimposed on lungs that had undergone resection of the right upper lobe and received radiation. In addition, more likely than not, he had persistent carcinoma in his lungs at the time of his death. ... Any mild airflow obstruction he had prior to the events after his lung cancer surgery, would not have contributed in any major way to his demise. His demise was related to lung compromise not consequent or hastened by his past coal mine employment.

* * *

In CONCLUSION, it can be stated with a reasonable degree of medical certainty, that Mr. Perkins did not have CWP or associated impairment. While he had some degree of airflow obstruction, this was related to his past smoking history; it was not disabling prior to his lung

cancer surgery. Towards the latter stages of his life, his smoking-related COPD in combination with his radiation pneumonitis, lung resection and probable persistent carcinoma, all contributed and caused his death. He also had disabilities related to heart disease, back problems, etc. These were not conditions related to the past inhalation of coal mine dust exposure. My opinions with respect to his impairments and disability would not change if he was found to have CWP.

EX-1.

Dr. James R. Castle

Dr. Castle conducted an extensive record evaluation on behalf of the employer. His consultation report, dated August 27, 2004, consists of 32 pages of his detailed analysis of pertinent documents. EX-2. Dr. Castle, who is board-certified in internal medicine and pulmonary disease, is a B-reader and has also been a Clinical Professor of Medicine at the University of Virginia since 1993. EX-2. As part of his medical practice, Dr. Castle sees over 20 patients per day, with a focus on pulmonary disease. EX-3 at 7.

Based on his review, Dr. Castle opined that the Miner did not suffer from coal workers' pneumoconiosis. He noted that Mr. Perkins's coal mine work history would be sufficient to develop pneumoconiosis. He also considered the Miner's cigarette smoking history to be a relevant risk factor as well. EX-2 at p. 29. With respect to the smoking history, Dr. Castle noted:

There was a somewhat variable history of tobacco smoking in this individual. His spouse indicated in the interrogatories that he had smoked three quarter packs of cigarettes daily for 29 years and stopped smoking in 1983. Hospital records indicated that he had smoked as much as one to two packs of cigarettes daily for as long as 33 years. Either of these histories is sufficient enough to have caused him to develop chronic obstructive pulmonary disease, i.e. chronic bronchitis/emphysema and/or lung cancer and/or atherosclerotic cardiovascular disease ...

Another risk factor for the development of pulmonary symptoms is that of cardiac disease. He did in fact develop coronary artery disease as documented by electrocardiograms, cardiac catheterizations, and other investigations. ... He also had documented evidence of a lung cancer, bronchoalveolar type, with metastases to lymph nodes. This resulted in his having a right upper lobectomy ... as well as postoperative radiation therapy. This treatment resulted in a significant scarring in that area with resultant chronic problems with clearance of secretions. ...

At no time did he demonstrate any consistent physical findings indicating the presence of a chronic interstitial pulmonary process such as

coal workers' pneumoconiosis. He did not have a consistent finding of rales, crackles, or crepitations. He did have the finding of rhonchi or wheezes on several occasions indicating the presence of chronic airway obstruction due to his tobacco smoking habit.

The vast majority of radiologists and B-readers felt that there was no evidence whatsoever of any form of pneumoconiosis radiographically. This was further confirmed by CT scans which did not show evidence of coal workers' pneumoconiosis. ...

The physiologic studies that were done were somewhat variable over time. While it was noted by some individuals that he had a restrictive pulmonary process, this was never documented by a reduction in total lung capacity. He did demonstrate a variable, significantly reversible airway obstruction. ... The degree of reversibility and the variability in the actual data over time indicates that he did have tobacco smoke induced chronic airway obstruction. These findings are not indicative of airway obstruction due to coal workers' pneumoconiosis. Therefore, it is my opinion that he did have a variable degree of airway obstruction between mild and moderate. This finding was due to his tobacco abuse.

* * *

Although an autopsy was not performed at the time of death, he did have pathologic specimens from resection lung surgery in 1996. Both Drs. Raphael Caffrey and Stephen Bush were unable to establish a diagnosis of coal workers' pneumoconiosis pathologically based upon these tissue reviews. Therefore, since the pathologic evidence is the "gold standard" for the diagnosis of this disease process, coal workers' pneumoconiosis was not present in this case. ...

Therefore, for the reasons stated above, it is my opinion with a reasonable degree of medical certainty that Mr. Moses Perkins did not suffer from coal workers' pneumoconiosis. He did not have the physical findings, the radiographic findings, the physiologic findings, the arterial blood gas findings, or the pathologic findings to indicate the presence of coal workers' pneumoconiosis.

It is my opinion with a reasonable degree of medical certainty that Mr. Perkins was not permanently and totally disabled during life as a result of coal workers' pneumoconiosis. ... [H]e was permanently and totally disabled from the pulmonary point of view due to tobacco smoke induced chronic obstructive airways disease and lung cancer. None of these conditions was caused by, aggravated by, or contributed to by coal workers' pneumoconiosis. ...

It is my opinion with a reasonable degree of medical certainty that his death was not caused by, contributed to, or hastened by coal workers' pneumoconiosis or any other process arising from his coal mine employment duties. His death was due to complications from lung cancer and tobacco smoke induced chronic obstructive pulmonary disease. It was also complicated by severe lung secretion clearance problems related to scarring from radiation therapy due to lung cancer. He had developed poly bacterial pneumonias requiring frequent bronchoscopies. Nevertheless, there is no evidence that coal workers' pneumoconiosis or his coal mine dust exposure played any role in his demise whatsoever. This is further corroborated by the fact that he did not have pathologic evidence of coal workers' pneumoconiosis.

EX-2.

CT & Perfusion Scan Evidence

A CT scan was conducted on June 22, 1992. DX-2 [EX-9 M]. Dr. Ahmed concluded that this scan demonstrated that

[n]o significant mediastinal masses noted. Some calcified lymph nodes are seen. Platelike/segmented atelectasis in the lingual of left lung scan with some pleural pericardial adhesions. CT scan is otherwise grossly unremarkable.

Dr. Wheeler read this CT scan on January 27, 1994, and concluded that it showed no pneumoconiosis. DX-2 [EX-1].

Dr. Wiot interpreted this CT scan on March 16, 1994, and opined that the scan was "within normal limits." DX-2 [EX-4].

Dr. Fishman's interpretation of this CT scan indicated that he found "very minimal fibrosis in the left base ... [and that] no other abnormalities were seen." DX-2 [EX-6].

A perfusion scan was conducted on November 4, 1999. DX-13. The study discovered a right upper lobe defect corresponding to post surgical and post radiation therapy changes in the right upper lobe[.]

A chest CT scan was conducted at the Princeton Community Hospital on November 15, 1999. DX-13. The "medial aspect of the right upper chest [had] extensive pleural and parenchymal scarring[.] ... The right upper chest [had] volume loss ... compatible with post surgical and post radiotherapy change." The "left lung [was] clear." The radiologist's impression was an unchanged "post surgical and post radiotherapy change in the right chest[.]" No new masses were discovered.

Deposition Testimony from Survivor's Claim

Dr. James R. Castle

Dr. Castle's deposition testimony was recorded on September 21, 2004. EX-3. The primary focus of this testimony was a review of his medical report, dated August 27, 2004 (EX-2), with added comments on the medical report by Dr. Rosenberg (EX-1).

Dr. Castle testified that the Miner was disabled by a respiratory impairment, and that he suffered from a "mild to moderate degree of very significant reversible airway obstruction without restriction but with gas trapping[.]" Dr. Castle reiterated that Mr. Perkins did not demonstrate a restrictive process. EX-3 at 11. He concluded that Mr. Perkins "had tobacco smoke-induced respiratory impairment initially that was associated, as I indicated, with a variable degree of significantly reversible obstruction and gas trapping which ultimately became complicated by lung cancer and the treatment therefrom." EX-3 at 12.

Dr. Castle was questioned about the results from biopsies that were conducted. He acknowledged that anthracotic pigments were detected in the Miner's lung. This was not pneumoconiosis, he emphasized, because "[a]nthracotic pigment simply means the presence of a black pigment which could be due to coal dust, but it could also be due to carbon from any number of different causes, including tobacco smoking." EX-3 at 15. He was also queried about the presence of "legal pneumoconiosis" and testified to his understanding of the statutory disease as broadly defined but concluded that "Mr. Perkins did not have that entity." EX-3 at 17. He also opined that Mr. Perkins's death occurred as a result of complications of lung cancer due to tobacco abuse, and that he would have died "at the same time regardless of his occupational history." Dr. Castle also emphasized that coal mine dust exposure does not cause lung cancer. EX-3 at 18-19.

On cross-examination, Dr. Castle emphasized that, assuming the Miner suffered from coal workers' pneumoconiosis, his opinion would remain the same. EX-3 at 19.

Dr. Stephen T. Bush

Dr. Bush's deposition testimony was recorded on September 23, 2004. EX-4. His testimony focused on his review of the results of eight biopsies and Mr. Perkins's medical records. He testified that the biopsies were conducted to determine the cause of the mass that was detected in the Miner's right upper lobe. EX-4 at 17.

He opined that the fibrosis and chronic inflammation discovered in a biopsy conducted in 1996 was due to lung cancer. EX-4 at 18. He explained that the type of cancer – adenocarcinoma – would invade tissues at the periphery of the primary growth and would stimulate scarring or fibrous reaction.

Dr. Bush was asked about his findings of silica and silicate birefringent particles. Despite these findings, he said, a diagnosis of silicosis would not be made in this case, because he did not find any associated scarring or fibrous reaction that would be characteristic of silicosis. EX-4 at 21-22. Similarly, Dr. Bush opined that his findings of black pigment would not entail a diagnosis of pneumoconiosis in this case. EX-4 at 22. He also testified that biopsies that were conducted after radiation therapy showed a fibrosis that may be a "radiation fibrosis." EX-4 at 24.

There is sufficient lung tissue available from which a diagnosis of pneumoconiosis could be made. The 1996 biopsy yielded numerous slides, and Dr. Bush concluded that the histological slides did not demonstrate the existence of pneumoconiosis. EX-4 at 27. He noted as well that the slides came from the right upper lobe, and further explained that it would be in the upper lobes where pneumoconiosis would typically be found. EX-4 at 27-28.

On cross-examination, Dr. Bush acknowledged that these biopsies, especially the needle biopsies, were conducted primarily to ascertain the presence of cancer. EX-4 at 31. He was also asked whether coal mine dust exposure can cause diseases other than clinical pneumoconiosis:

Coal workers' pneumoconiosis can cause emphysema of several types. It can cause chronic bronchitis. These conditions may produce some degree of symptoms. The emphysema that is resulting from coal mine dust exposure, in general, parallels the degree of pneumoconiosis present. In other words, when there is severe pneumoconiosis, you may often find a fairly large amount of emphysema and vice versa.

EX-4 at 33. He also acknowledged that "mine dust emphysema" may be present without clinical pneumoconiosis. He also said that the silica particles and black pigment would likely have resulted from the Miner's coal mine dust exposure. Yet they did not prompt a fibrotic reaction, and these conditions alone would not constitute pneumoconiosis. EX-4 at 34-35. He added on redirect examination that these conditions were not enough to have caused the Miner's chronic obstructive pulmonary disease. EX-4 at 35. He also had found no focal emphysema.

Opinion Evidence from the Miner's Claim

West Virginia Occupational Pneumoconiosis Board

West Virginia Occupational Pneumoconiosis Board on October 1, 1985 awarded the Miner with a 20% award. DX-2.

Dr. D. L. Rasmussen

Dr. Rasmussen conducted a thorough examination of the Miner on June 28, 1993 at the request of the Department of Labor. DX-2 [DX-11]. He recorded a 33 year

history of smoking at the rate of 1/2 to 3/4 pack per day. Relying on physical examination, a chest x-ray read as positive by Dr. Manu Patel, and a battery of clinical tests, including a ventilatory test that showed an irreversible impairment, Dr. Rasmussen diagnosed pneumoconiosis based on 22 years of coal mine employment and the positive x-ray. He also diagnosed "legal" pneumoconiosis, viz. chronic bronchitis based on the chronic productive cough and "ASHD – Myocardial infarction in 1991." He remarked that the "two risk factors" in the etiology of the pulmonary diagnoses were coal mine dust exposure and cigarette smoking, of which the former was "a major contributing factor."

Dr. Rasmussen is board-certified in internal medicine, board-eligible in pulmonary disease, and had extensive clinical experience. *See Martin v. Ligon Preparation Co.*, 400 F.3d 302, 307, (6th Cir. 2005).

Dr. Patel, as noted above, interpreted the June 28, 1993 chest x-ray as positive ("1/0"). This film formed part of the basis of Dr. Rasmussen's diagnosis of clinical pneumoconiosis. DX-2 [DX-14]. Dr. Patel is board-certified in radiology. This film was reread on August 24, 1983, as negative by Dr. Paul Franke, a board-certified radiologist and B-reader. DX-2 [DX-15].

Dr. J. Randolph Forehand

Dr. Forehand examined the Miner on February 23, 1994, and reported on his evaluation the following day. DX-2 [CX-1]. Mr. Perkins told him that he had smoked cigarettes for 31 years until 1980 at the rate of 1/2 pack per day. Dr. Forehand read a chest x-ray with the ILO classification of "0/1." He diagnosed, *inter alia*, "COPD" which he attributed to coal mine dust exposure. Dr. Forehand relies in part on epidemiological studies to demonstrate that coal mine dust exposure would have an aggravating effect on the Miner's COPD. The Miner's "disabling symptoms" were attributed in part to coal mine dust exposure. Dr. Forehand is board-certified in pediatrics and Allergy and Immunology.

Dr. Shawn A. Chillag DX-2 [EX-22]

Dr. Chillag submitted a report on July 8, 1994 following his evaluation of the Miner's medical records. He opined that the Miner did not suffer from coal workers' pneumoconiosis. Mr. Perkins did suffer from a pulmonary impairment, but Dr. Chillag attributed that impairment to the Miner's smoking, and further opined that the Miner's disability was due to recent myocardial infarction and angioplasty, with minor contributing factors from seizure disorder, pulmonary impairment, back injury and fractures. In a letter dated July 22, 1994, he stated that the Miner did not have any respiratory or pulmonary impairment significantly related to or substantially aggravated by coal mine dust exposure.

Dr. Gregory J. Fino

Dr. Fino reviewed the Miner's medical records, and reported on this evaluation on July 20, 1994. DX-2 Dr. Fino noted that the majority of x-ray readings that were before him were negative. He also felt that Mr. Perkins did not have pneumoconiosis based on the improvement in his pulmonary function results. Dr. Fino explained that the ventilatory tests showed a pure obstructive defect, with no restrictive defect, and that the tests showed an "improvement in the small airways." The proportionate reduction in the small airway flow over the corresponding reduction in large airway flow "is not consistent with a coal dust related condition but is consistent with conditions such as cigarette smoking, pulmonary emphysema, non-occupational chronic bronchitis, and asthma."

Dr. Fino emphasized that the pattern would not be consistent with a coal dust related condition, but instead a pure obstructive ventilatory abnormality seen in asthma or in "conditions related to cigarette smoking." He also explained that the lung volumes were consistent with over-inflation, and not the under-inflation due to contraction due to fibrotic scarring.

Dr. Fino thought that the Miner did not have simple pneumoconiosis or an "occupationally acquired pulmonary condition."

Dr. W. K. C. Morgan

Dr. Morgan conducted an extensive review of Mr. Perkins's medical records. He opined that the Miner did not suffer from pneumoconiosis. He remarked on the smoking history, which was variably reported, noting that Mr. Perkins's smoking history appeared to Dr. Morgan to be more extensive prior to the Miner's claim for black lung benefits. Dr. Morgan likewise thought that some of the positive x-ray interpretations may have been influenced by a black lung claim. A CT scan was read as negative, even by one of the radiologists who had offered a positive reading for the Miner. Based on this, Dr. Morgan opined that the Miner did not have pneumoconiosis, but a "naturally occurring" obstructive disease significantly derived from the Miner's smoking.

Dr. Peter G. Tuteur

Dr. Tuteur submitted a consultation report on July 20, 1994, in which he concluded that the Miner did not suffer from pneumoconiosis. There was "insufficient objective evidence" to justify a diagnosis of "clinically-significant ..." coal workers' pneumoconiosis.

Dr. Bruce N. Stewart

Dr. Stewart evaluated Mr. Perkins and submitted his report on January 20, 1994. DX-2 [EX-3]. He recorded a history of smoking 1/2 pack of cigarettes per day from age 18 until 1980. He diagnosed, *inter alia*, chronic bronchitis. Dr. Stewart also concluded

that the Miner did not suffer from coal workers' pneumoconiosis. He also opined that Mr. Perkins suffered from a pulmonary or respiratory impairment, indeed, that he was totally disabled, but attributed that to his smoking history of 25-33 years. Dr. Stewart noted that the Miner had a history of a productive cough "despite the fact that he ha[d] not been in the mines since 1977. This would eliminate industrial bronchitis as a cause for the cough."

According to Dr. Stewart:

The etiology of this total respiratory impairment, however, is explained by the combination of factors including ischemic heart disease, compression fracture of the thoracic spine, chronic bronchitis from smoking cigarettes, and weakened left hemi-diaphragm. None of these diagnoses, however, are caused in whole or in part by ventilation of coal dust or coal workers' pneumoconiosis.

DX-2 [EX-3].

DISCUSSION

Entitlement in Survivor's Claim

The provisions at 20 C.F.R. § 718.205 require competent medical evidence, which (1) establishes that the miner died due to pneumoconiosis; or (2) that pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or the death was caused by complications of pneumoconiosis; or (3) that the presumption of 20 C.F.R. § 718.304 is applicable. Pneumoconiosis constitutes a "substantially contributing cause" if it serves to hasten death in any way. 20 C.F.R. § 718.205(c)(5). See *Richardson v. Director, OWCP*, 94 F.3d 164, 167, 21 B.L.R. 2-373 (4th Cir.1996); *Shuff v. Cedar Coal Co.*, 967 F.2d 977, 979-80, 16 B.L.R. 2-90 (4th Cir. 1992), *cert. denied* 506 U.S. 1050 (1993). See *Consolidation Coal Co. v. Kramer*, 305 F.3d 203, 205, 22 B.L.R. 2-469 (3d Cir. 2002) (applying Fourth Circuit law); *Lukosevicz v. Director, OWCP*, 888 F.2d 1001, 1003, 13 B.L.R. 2-100 (3d Cir. 1989). While an award may be cognizable if pneumoconiosis contributes to the miner's death, albeit briefly, see *Northern Coal Co. v. Director, OWCP*, 100 F.3d 871, 874, 20 B.L.R. 2-335 (10th Cir. 1996), the standard is not satisfied if pneumoconiosis contributed to the miner's death to a "negligible" degree. See *Grizzle v. Pickands Mather & Co.*, 994 F.2d 1093, 1095, 17 B.L.R. 2-123 (4th Cir. 1993).

Existence of Pneumoconiosis

In a survivor's claim under Part 718, the administrative law judge must normally make a threshold determination as to the existence of pneumoconiosis under 20 C.F.R. § 718.202(a) prior to considering whether the Miner's death was due to pneumoconiosis. *Trumbo v. Reading Anthracite Co.*, 17 B.L.R. 1-85 (1993). The existence of pneumoconiosis may be established by any one or more of the following

methods: (1) chest x-rays; (2) autopsy or biopsy; (3) by operation of presumption; or (4) by a physician exercising sound medical judgment based on objective medical evidence. 20 C.F.R. § 718.202(a). Because this claim arises within the territorial jurisdiction of the United States Court of Appeals for the Fourth Circuit, the adjudicator must weigh all of the evidence together in reaching a finding as to whether a miner has established that he has pneumoconiosis. *Island Creek Coal Co. v. Compton*, 211 F.3d 203, 211, 22 B.L.R. 2-162 (4th Cir. 2000). See *Penn Allegheny Coal Co. v. Williams*, 114 F.3d 22, 21 B.L.R. 2-104 (3rd Cir. 1997).

Pneumoconiosis under the Act is defined as both clinical pneumoconiosis and/or any respiratory or pulmonary condition significantly related to or significantly aggravated by coal dust exposure:

For the purpose of the Act, “pneumoconiosis” means a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. This definition includes, but is not limited to, coal workers’ pneumoconiosis, anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, progressive massive fibrosis, silicosis or silico-tuberculosis, arising out of coal mine employment. For purposes of this definition, a disease “arising out of coal mine employment” includes any chronic pulmonary disease resulting in respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment.

20 C.F.R. § 718.201.

20 C.F.R. § 718.202(a)(1)

The first method by which a claimant can demonstrate the existence of pneumoconiosis is by x-ray evidence at Section 718.202(a)(1).

The regulation at 20 C.F.R. § 718.202(a)(1) requires that “where two or more X-ray reports are in conflict, in evaluating such X-ray reports consideration shall be given to the radiological qualifications of the physicians interpreting such X-rays.”² In this vein, the Board has held that it is proper to accord greater weight to the interpretation of a B-reader or Board-certified radiologist over that of a physician without these specialized qualifications. *Roberts v. Bethlehem Mines Corp.*, 8 B.L.R. 1-211 (1985); *Allen v. Riley Hall Coal Co.*, 6 B.L.R. 1-376 (1983). Moreover, an interpretation by a dually-qualified B-reader and Board-certified radiologist may be accorded greater weight than that of a B-reader. See *Zeigler Coal Co. v. Director, OWCP [Hawker]*, 326 F.3d

² A “B-reader” (B) is a physician, but not necessarily a radiologist, who successfully completed an examination in interpreting x-ray studies conducted by, or on behalf of, the Appalachian Laboratory for Occupational Safety and Health (ALOSH). A designation of “Board-certified” (BCR) denotes a physician who has been certified in radiology or diagnostic roentgenology by the American Board of Radiology or the American Osteopathic Association.

894, 899 (7th Cir. 2003). Finally, a radiologist's academic teaching credentials in the field of radiology are relevant to the evaluation of the weight to be assigned to that expert's conclusions. See *Worhach v. Director, OWCP*, 17 B.L.R. 1-105 (1993). I emphasize, however, that the adjudicator is not required to defer to the interpretations by a radiologist who holds an academic position or professorship. See *Chaffin v. Peter Cave Coal Co.*, 22 B.L.R. 1-294 (2003). The party seeking to rely on an x-ray interpretation bears the burden of establishing the qualifications of the reader. *Rankin v. Keystone Coal Mining Co.*, 8 B.L.R. 1-54 (1985).

Upon review of the chest x-rays that have been submitted with the survivor's claim, as well as the extensive x-ray evidence from the Miner's claim, I find that Claimant has failed to demonstrate on the basis of the x-ray evidence that the Miner suffered from pneumoconiosis. The preponderance of the x-ray interpretations does not support a finding of pneumoconiosis. Indeed, in the Miner's claim, the positive readings by the Miner's experts, most of whom were dually qualified, were nevertheless matched by a great number of negative interpretations by similarly qualified radiologists, some of whom, including Dr. Wiot, possessed academic credentials as well. See *Worhach*.

I duly note that an adjudicator must not rely merely on the numerical superiority of x-ray interpretations. An administrative law judge is not required to defer to the numerical superiority of x-ray evidence. *Wilt v. Wolverine Mining Co.*, 14 B.L.R. 1-70 (1990). See also *Tokarcik v. Consolidation Coal Co.*, 6 B.L.R. 1-666 (1984). Moreover, the adjudicator should not blindly defer to later x-rays. See *Adkins v. Director, OWCP*, 958 F.2d 49, 16 B.L.R. 2-61 (4th Cir. 1992). I am also mindful that the Miner had secured numerous positive x-ray interpretations from radiologists who were both board-certified radiologists and B-readers.

Nevertheless, I find that the positive x-ray evidence that the Miner suffered from pneumoconiosis does not overcome the negative rereadings by similarly credentialed experts who interpreted the films on behalf of the employer. See generally *Napier v. Director, OWCP*, 890 F.2d 669, 671, 13 B.L.R. 2-117 (4th Cir. 1989) (rational basis for ALJ to resolve conflicting interpretations of x-rays by deferring to rereadings by B-readers); *Edmiston v. F&R Coal Co.* 14 B.L.R. 1-65 (1990). Moreover, I credit the negative rereadings of recent films, dated May 5 and July 31, 2001, by Dr. Wiot, on the basis of his superior credentials. Even discounting his interpretations, I note that the Claimant has offered the opinion that these specific x-rays are not sufficient for determining the presence of coal workers' pneumoconiosis. The array of recent films taken during the last half-year of the Miner's life during treatment at the Princeton Hospital are unclassified, and do not purport to show that he suffered from coal workers' pneumoconiosis.

I note that two x-rays dated May 5, 2001 and July 31, 2002, reread by Dr. Wiot and in the record at DX 24, are digital x-rays. See *Webber v. Peabody Coal Co.*, 23 B.L.R. 1-___, BRB No. 05-0335 BLA (Jan. 27, 2006)(en banc). In *Webber*, the BRB held that digital x-ray interpretations are not considered chest x-ray evidence under 20 C.F.R. §§ 718.101(b), 718.102, 718.202(a)(1), and Appendix A. As a result, the Board

held that digital chest x-rays are properly considered under 20 C.F.R. § 718.107 only when the administrative law judge determines, on a case-by-case basis pursuant to 20 C.F.R. § 718.107(b), that the proponent of the digital x-ray evidence has established that it is medically acceptable and relevant to entitlement. In this case, Dr. Wiot states at DX 24 that these digital films are of good quality and acceptable by ILO standards. With Dr. Wiot's distinguished qualifications, I will consider these digital x-rays in deciding this case.

In the final analysis, taking a qualitative, as well as quantitative, approach to the vast radiographic evidence, I am unable to find that it is more likely than not that Mr. Perkins suffered from pneumoconiosis on the basis of the x-ray evidence as a whole. *See Woodward v. Director, OWCP*, 991 F.2d 314, 321, 17 B.L.R. 2-77 (6th Cir. 1993). At the most, I find that the chest x-ray evidence as a whole is equally probative, and does not demonstrate that it is more likely than not that the Miner suffered from coal workers' pneumoconiosis.

20 C.F.R. § 718.202(a)(2)

A Claimant may establish the presence of pneumoconiosis at Section 718.202(a)(2), upon the basis of autopsy or biopsy evidence. There have been a number of biopsies in this case. I find that the Claimant has not demonstrated the existence of pneumoconiosis on the basis of biopsy evidence, however. I find most persuasive the analysis of the histologic evidence by Dr. Bush, who reviewed the pathology specimens and histological slides, and offered a comprehensive report based on his study. Although anthracotic pigment and evidence of silica and silicate birefringent particles were present, and they may have been derived from coal mine dust exposure, Dr. Bush reasonably opined that the amount of this pigmentation did not entail a finding of pneumoconiosis. Nor would the silica and silicate birefringent particles found in the 1996 slides constitute pneumoconiosis, he explained, because these findings were not accompanied by any associated scarring or fibrous reaction that would be characteristic of silicosis. Finally, I credit Dr. Bush's opinion that the findings in the biopsy slides did not support findings of emphysema or chronic obstructive pulmonary disease derived from coal mine dust exposure.

Based on the thorough explanation of Dr. Bush's findings, as well as his credentials, and his finding that the biopsy evidence as a whole does not demonstrate the existence of pneumoconiosis, I find that the Claimant has not demonstrated the existence of coal workers' pneumoconiosis on the basis of the biopsy evidence of record.

20 C.F.R. § 718.202(a)(3)

Section 718.202(a)(3) provides that pneumoconiosis may be established if any one of several cited presumptions applies. This provision is unavailable to this Claimant. In this case, the presumption of Section 718.304 does not apply because there is no evidence in the record of complicated pneumoconiosis; Section 718.305 is

not applicable to claims filed after January 1, 1982. Finally, the presumption of § 718.306 is applicable only in a survivor's claim filed prior to June 30, 1982.

20 C.F.R. § 718.202(a)(4)

The Claimant can also demonstrate the existence of pneumoconiosis on the basis of medical opinion evidence. 20 C.F.R. § 718.202(a)(4). A determination of the existence of pneumoconiosis may be made, notwithstanding a negative x-ray, if a physician, exercising sound medical judgment finds that the miner suffers from pneumoconiosis as defined in 20 C.F.R. § 718.201. Any such finding shall be based on objective medical evidence, such as arterial blood gas tests, physical performance tests, physical examination, and medical and work histories. Such a finding shall be supported by a reasoned medical opinion.

It is well-established that pneumoconiosis is expansively defined in the Act, such that an obstructive pulmonary or respiratory impairment may constitute statutory pneumoconiosis, *Warth v. Southern Ohio Coal Co.*, 60 F.3d 173, 175, 19 B.L.R. 2-265 (4th Cir. 1995). See also *Freeman United Coal Mining Co. v. Summers*, 272 F.3d 473, 481, 22 B.L.R. 2-265 (7th Cir. 2001); *Eagle v. Armco Inc.*, 943 F.2d 509, 511 n.2, 15 B.L.R. 2-201 (4th Cir. 1991); *Old Ben Coal Co. v. Prewitt*, 755 F.2d 588, 591 (7th Cir. 1985) (chronic obstructive pulmonary disease meets statutory definition whether or not technical pneumoconiosis), *provided* the pulmonary or respiratory disease is significantly related to or substantially aggravated by the Miner's coal mine dust exposure. See 20 C.F.R. §718.201; *Stiltner v. Island Creek Coal Co.*, 86 F.3d 337, 341, 20 B.L.R. 2-246 (4th Cir. 1996). See generally 65 Fed. Reg. 79943 (Dec. 20, 2000) (citing cases).

With this in mind, I will accord less weight to the medical opinions of Drs. Stewart, Chillag, Tuteur and Morgan that were submitted in the Miner's claim to the extent they do not adequately account for the expansive definition of "legal" coal workers' pneumoconiosis. I do credit their views that the Miner did not suffer from "clinical" pneumoconiosis. I duly note, however, Dr. Tuteur's observation in his deposition testimony that variability in clinical test results without restriction would not be consistent with CWP. DX-2 [EX-25].

I have carefully reviewed the medical opinions of Drs. Rasmussen and Forehand. Both physicians had attributed Mr. Perkins's chronic obstructive pulmonary disease at least in part to coal mine dust exposure.

In the case of the former, Dr. Rasmussen thought that coal mine dust exposure and smoking were two risk factors that played a role in the Miner's pulmonary impairment. I must discount his diagnosis of pneumoconiosis, which he considers to have been "based on 22 years of coal mine employment and the positive x-ray." That x-ray, while read as positive by Dr. Patel, a board-certified radiologist, was reread as negative by Dr. Franke, who is a dually qualified radiologist and B-reader. I credit the negative rereading. See *Roberts*. I find that the negative rereading of the x-ray on

which Dr. Rasmussen relies undermines to some extent the documentary support for his diagnosis of pneumoconiosis. See *Winters v. Director, OWCP*, 6 B.L.R. 1-877 (1984). While a medical opinion diagnosis of pneumoconiosis may be sufficient notwithstanding a negative x-ray, see *Taylor v. Director, OWCP*, 9 B.L.R. 1-22 (1996), where x-ray evidence constitutes a major part of the physician's documentation, his opinion may be entitled to diminished probative weight if *that* film has been reread as negative. Cf. *Director, OWCP v. Rowe*, 710 F.2d 251, 255 n. 6, 5 B.L.R. 2-99 (6th Cir. 1983) (validity of opinion discounted because doctor relied on x-ray found to be unreadable). I hasten to note that Dr. Rasmussen's diagnosis is not confined to clinical pneumoconiosis, and that his attribution of the Miner's COPD most to coal mine dust exposure qualifies as pneumoconiosis.

Although diagnoses of coal workers' pneumoconiosis occasionally show up in the medical records upon which the Claimant relies, and the hospital records contain many references to chronic obstructive pulmonary disease, I do not find medical opinion evidence in these records that persuasively ties any pulmonary or respiratory condition to Mr. Perkins's coal mine dust exposure.

I shall address the death certificate at this juncture. The Claimant places considerable reliance on this document, certified as it is by Dr. Patel, who was one of the Miner's treating physicians. I consider this certificate to be entitled to little weight to the extent Dr. Patel certifies death in part due to pneumoconiosis. Although its conclusions are consistent with clinical findings in the record of the Miner's hospitalizations and treatment from Dr. Patel to the extent he observed and diagnosed chronic obstructive lung disease and at times noted pneumoconiosis, I do not see a reasonable basis from the hospitalization and treatment notes that this pulmonary or respiratory impairment would constitute pneumoconiosis. As such, the death certificate suffers from a lack of sound documentation. The Claimant also relies on the clinical records of the Miner's hospitalizations and treatment. Yet there is no convincing expert conclusion in the principal exhibits offered on behalf of the claim, DX-13 to DX-15, that ties Mr. Perkins's chronic obstructive pulmonary disease or lung disease to his coal mine dust exposure. To the extent the death certificate relies on the documentation from the hospitalization and treatment records, I find that there is insufficient support for the certificate's conclusions in the record. See *Smakula v. Weinberger*, 572 F.2d 127, 131-32 (3d Cir. 1978).

In the final analysis, taking into account the "qualifications of the respective physicians, the explanations of their medical opinions, the documentation underlying their medical judgments and the sophistication and bases of their diagnoses," see *Sterling Smokeless Coal Co. v. Akers*, 131 F.3d 438, 441, 21 B.L.R. 2-269 (4th Cir. 1997), I am most persuaded by the opinions by Dr. Rosenberg and Castle. Their opinions with respect to this issue are better documented and explained, and make better sense in light of the record. See generally *Clark v. Karst-Robbins Corp.*, 12 B.L.R. 1-149 (1989) (*en banc*); *Lucostic v. United States Steel Corp.*, 8 B.L.R. 1-46 (1985).

Each doctor offers persuasive reviews of the medical record, and both of their opinions with respect to “legal” pneumoconiosis are detailed and well documented. Dr. Castle, for example, points out the degree of variability and reversibility in the evidence that militates against a finding of a coal mine dust induced chronic obstructive pulmonary impairment, or an airway obstruction due to pneumoconiosis. He cited the absence of “consistent findings of rales, crackles, or crepitations” that would suggest a chronic interstitial pulmonary process such as coal workers’ pneumoconiosis. Dr. Castle also noted that findings of rhonchi and wheezes would suggest a smoking-induced chronic airway obstruction. Dr. Rosenberg noted that ventilatory tests showed a variable obstruction, that the studies did not show a restriction but did evidence to some extent air trapping. While acknowledging that coal mine dust exposure can cause airflow obstruction, Dr. Rosenberg’s explanation that the pattern of airflow obstruction as shown by air trapping was more suggestive of a smoking derived obstruction. Dr. Fino cited the lung volume results that were, in his view, consistent with an over-inflation condition that would not indicate fibrotic scarring as well as airflow results that were thought not to be consistent with a coal mine dust related condition.

At the end of the day, the analyses of the record by Drs. Rosenberg, Castle and Fino, and to a lesser extent the Employer’s other experts who submitted reports for the Miner’s claim, are far more detailed, documented and reasoned than the competing opinions of Drs. Forehand, Rasmussen, Jarboe and Patel. Similarly, while diagnoses of COPD and, occasionally, pneumoconiosis, appear in the vast treatment records, there is nothing to suggest that these descriptions of the Miner’s illness are thought out. Indeed, these diagnoses appear to be “carried” forward as Mr. Perkins presented for treatment from time to time.

Drs. Castle and Rosenberg also possess superior credentials in the relevant fields than do Drs. Rasmussen and Forehand. Although Dr. Rasmussen’s and Dr. Forehand’s extensive clinical experience is important and has been taken into account, see *Martin v. Ligon Preparation Co.*, 400 F.3d at 307, I note that both Drs. Castle and Rosenberg have academic credentials that bolster the credibility of their opinions. See *Worhach*. In addition, they have a more accurate picture of the Miner’s health, having examined a greater amount of medical documentation that was developed in the years since the Miner had been seen by Drs. Rasmussen and Forehand. Cf. *Balsavage v. Director, OWCP*, 295 F.3d 390, 397, 22 B.L.R. 2-386 (3d Cir. 2002) (opinion of physician who did not address other medical records accorded less weight).

Finally, the CT scan evidence does not show the presence of either clinical or a pulmonary or respiratory impairment significantly related to or substantially aggravated by the Miner’s coal mine dust exposure. Even viewing this evidence with caution, see *Consolidation Coal Co. v. Director, OWCP [Stein]*, 294 F.3d 885, 892-93, 22 B.L.R. 2-409 (7th Cir. 2002) (negative CT scan does not rule out legal pneumoconiosis), I find that this evidence further militates against a demonstration of pneumoconiosis at Section 718.202(a)(4).

Taking this evidence into account, and reviewing as well the evidence from the Miner's claim *de novo*, I find that the Claimant has not demonstrated the existence of pneumoconiosis at Section 718.202(a)(4).

Finally, I must evaluate all relevant evidence to determine whether the Claimant has established that Mr. Perkins suffered from the disease. *Compton*. On this record, after weighing all relevant evidence, I find that the Miner did not suffer from pneumoconiosis. None of the relevant provisions of Section 718.202(a) have been satisfied. When analyzed together, this evidence does not establish the presence of coal workers' pneumoconiosis.

Because the Claimant has not established that the Miner suffered from either clinical pneumoconiosis, or any pulmonary or respiratory impairment significantly related to, or substantially aggravated by, the Miner's coal mine dust exposure, I must find that Mrs. Perkins does not qualify for benefits under the Act. *Trumbo*.

Death due to Pneumoconiosis

Even assuming that Mr. Perkins suffered from pneumoconiosis, I find that the record does not establish that his death was hastened, even by a minimal degree, by pneumoconiosis. Certainly, the presence of coal workers' pneumoconiosis may be shown to have hastened death if it is demonstrated to have precluded treatment or to have compromised the Miner's health so that he succumbed to his other conditions. This conclusion, if credited, would constitute an adequate rationale for the opinion that pneumoconiosis hastened the Miner's death, albeit to a slight degree. See *Zeigler Coal Co. v. Director, OWCP [Villain]*, 312 F.3d 332, 334 (7th Cir. 2002). But no persuasive conclusion to that effect is offered in this case.

Assuming that the Miner suffered from pneumoconiosis, no physician has adequately "explain[ed] how ... [Mr. Perkins's] pneumoconiosis hastened his death." *Bill Branch Coal Corp. v. Sparks*, 213 F.3d 186, 192, 22 B.L.R. 2-251 (4th Cir. 2000); see *Freeman United Coal Mining Co. v. Cooper*, 965 F.2d 443, 450, 16 B.L.R. 2-74 (7th Cir. 1992) (treating physician's diagnosis of pneumoconiosis nevertheless does not establish death causation because conclusions are unexplained). The treatment notes, upon which the Claimant relies in an attempt to prove it more likely than not that pneumoconiosis hastened the Miner's death, even to a near *de minimis* degree, do not meet the Claimant's burden of proof.

Because the contrary opinions of the Employer's experts prevail in this case, I credit their view that pneumoconiosis did not hasten Mr. Perkins's death in any way or to any degree. *Grizzle*. Notwithstanding the death certificate, there is no convincing opinion that pneumoconiosis hastened the Miner's death. The medical records from the final months of Mr. Perkins's life establish instead the effects of his lung cancer and the scarring and infection that was derived from radiation treatment.

In reaching this conclusion, I have accounted for the fact that Dr. Patel was one of the Miner's treating physicians. Factors to be considered in weighing evidence from treating physicians include the nature and duration of the relationship, and the frequency and extent of treatment. In appropriate cases, a treating physician's opinion may be given controlling weight, provided that the decision to do so is based on the credibility of the opinion "in light of its reasoning and documentation, other relevant evidence and the record as a whole." 20 C.F.R. § 718.104(d) (2004). In the final analysis, the credibility of the treating physician's opinion may primarily rest on its "power to persuade." *Eastover Mining Co. v. Williams*, 338 F.3d 501, 513, 22 B.L.R. 2-625 (6th Cir. 2003). See *Jerico Mining, Inc. v. Napier*, 301 F.3d 703, 709, 22 B.L.R. 2-537 (6th Cir. 2002) (tribunal to examine opinions on their merits).

CONCLUSION

Because the Claimant has not established that the Miner's death was due to pneumoconiosis, or that pneumoconiosis hastened the Miner's death, I find that she has not established entitlement to survivor's benefits under the Act.

ATTORNEY'S FEES

The award of an attorney's fee under the Act is permitted only in cases in which Claimant is found entitled to benefits. Since benefits are not awarded in this case, the Act prohibits the charging of attorney's fees to the Claimant for representation services rendered in pursuit of the claim.

ORDER

The claim of Hazel L. Perkins for survivor's benefits under the Act is denied.

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WILLIAM S. COLWELL
Administrative Law Judge

Washington, D.C.

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. See 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used.

See 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed. At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Donald S. Shire, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. See 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).